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OUTLINE OF LECTURES

IN

SPECIAL PATHOLOGY

DEPARTMENT OF

Comparative Pathology, Bacteriology
and Meat Inspection

NEW YORK STATE VETERINARY COLLEGE
AT CORNELL UNIVERSITY, ITHACA, N. Y.

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INFLAMMATION.

Adami's conception of inflammation.

> "It is the series of changes constituting the local manifestation of the attempt at repair of actual or referred injury to a part, or, briefly, as the local attempt at repair of actual or referred injury."

Reaction to injury in lower animals (Invertebrates).

In Amoeba (unicellular organisms) the reactive process is twofold

(a) destruction or removal of irritant,—destruction by intracellular digestion, removal by extrusion of irritant.

(b) new growth of the organism.

This response is essentially reparative.

In multicellular organisms there is a separation of functions. Twofold reaction is clearly marked.

(a) destruction or removal of irritant is *in the main* accomplished by the wandering cells of mesoblastic origin.

(b) the new growth of tissue to replace that destroyed by the irritant proceeds *in the main* from the fixed cells of the tissue.

In higher forms of Metazoa, further differentiation of function among wandering cells,—more than one kind of leucocyte, some phagocytes, others not. Certain fixed cells especially endothelial cells of vessels have power of removing harmful matter.

In those in which a closed vascular system is not present, at first the attraction of leucocytes is the only recognizable reaction, proliferation of fixed cells later.

In higher metazoa with well developed vascular system, leucocytes collect at region of irritation.

Vascular phenomenon serves a twofold purpose:

(a) pouring out of increased fluid into injured area and

(b) collection and diapedesis of leucocytes.

Even in highest metazoa response to injury in a non vascular area, as cornea, may be with no change in neighboring blood vessels, but consist of a collection of leucocytes that were already in the injured area.

Determination of leucocytes to area of irritation is most constant early response to injury—proliferation is the prominent characteristic of later stages of the process.

Vascular changes as second and important adjuvant factor of later development (not in early metazoa).

Inflammatory reaction in non vascular tissue;—

mild irritants on cornea—destruction of cells followed by swelling and multiplication to make good the loss. No vascular changes. This is not common.

more intense irritation, e.g. corneal vaccinia, many wandering cells gather, remove foreign matter and protect proliferating corneal cells.

Phenomena, inflammatory reaction in non-vascular tissue.

(1) irritation and degeneration or death of cells,
(2) accumulation of wandering cells and
(3) regeneration of fixed tissue cells.

Inflammation in vascular tissues.

(1) Primary union of incised wounds, very slight vascular disturbance, hemorrhage slight, ends of vessels promptly closed by clots, very few tissue cells destroyed, slight fibrinous coagula glues edges together, at end of an hour wound appears closed and only slight reddening about incision, moderate hyperemia, conn. tissue cells send long processes into coagulum.
(2) Secondary union. Marked vascular reaction, exudative inflammation,
(3) Intense vascular reaction, suppurative inflammation, healing by granulation.
(4) Inflam. reaction with local death of tissue; necrotic inflammation.
(5) Inflam. reaction with diffuse death of tissue; diphtheritic inflammation.
(6) gangrenous inflammation.
(7) Invasion of blood stream by bacteria.

Inflammatory process.

Degeneration or death of tissues (initial injury).
Changes in the circulation.
- Active hyperemia,
- Exudation of blood plasma,
- Diapedesis of red corpuscles,
- Emigration of leucocytes,
  - Leucocytosis.
    - Polynuclear leucocytosis,
    - Mononucleosis—(typhoid, tuberculosis),
    - Eosinophilia—(parasites, skin diseases),
  - Lymphocytosis.

Changes in tissue cells in inflammation

- degeneration
  - disturbance of nutrition of cell, accumulation of intracellular secretion products (bile, glycogen), disturbance of specific cell ferments, autolysis,
  - action of exogenous ferments,
  - varieties : granular, hydropic, fatty,
  - mucous, glycogenic, hyaline, amyloid.
- necrosis
  - coagulation,
  - liquefaction,
  - caseous.
- regeneration of cells.

Variation in appearances in inflammation.

A. Nature of tissue affected

vascular and non vascular, loose and dense tissue.

e. g. cutaneous surfaces, exudation marked, vesicles and blisters.

mucous and cutaneous surfaces, tendency to remain localized, does not spread from surface so much.

serous surfaces, tendency to spread superficially because irritant carried into cavity affects other parts of serosa.
excretory organs suffer in removing irritating material—parenchymatous inflammation.

B. Position of tissues.

e. g. suppuration in deep and superficial tissue—abscess and ulcer, though process same, may be more serous exudate in ulcer ; more exudate near surface as a rule.

Peyer's patches become infiltrated and necrotic, but are cast off leaving an ulcer ; but the mesenteric glands, though similarly affected generally recover.

C. The relative intensity of the irritant is a greater cause of variation.

The ratio between virulence or intensity of irritant and resisting powers of the body.

e. g. heat slight produces redness, congestion
heat moderate " blisters
heat greater " death of tissue, etc.
strong chemical, death of few cells, prompt repair to deep necrosis, swelling about edges, pain, granulation tissue—scar.

- with pathogenic bacteria e. g. anthrax
  - very much attenuated—no recognizable effect;
  - not so much attenuated—some hyperemia, some infiltration of leucocytes, little fluid exudate, in day or two all trace gone;
  - slightly more virulent—migration of leucocytes—necrosis of tissue with abscess formation;
  - more virulent—no migration of leucocytes, exudation extends, leads to bacteremia.
- Same kind of change with same virus but using animals more and more susceptible.

D. The nature of the irritant.

- Can not classify inflams. according to nature of the irritant. no sharp line between those caused by heat and chemicals from those caused by bacteria for example.
- Suppuration may be produced not only by micro-organisms but also by chemicals—turpentine, croton oil, copper, silver nitrate, may produce suppuration.
- Under ordinary conditions suppuration indicates presence and growth of microbes.
- Is not common to all microbes.
- Pyogenic bacteria.
- Same microbe may produce different effects under different conditions.
- Classification of bacterial irritants (Adami, p. 210)

Other factors

- Duration of action, in general mechanical injury sets up simplest forms of inflammation.

Classification of inflammation. Nomenclature (Ewing) p. 50 sq.

1. Degeneration
2. Exudative inflammation
   - serous
   - fibrinous or croupous
   - hemorrhagic
   - purulent
3. Necrotic inflammation

Simple	abscess formation ulceration
Diphtheritic	exudative process with rapid and diffuse necrosis of superficial tissues.
Gangrenous	Large masses of tissue destroyed with decomposition.

4. Productive inflammation
    - (a) simple acute productive inflam.
        (cellular inflam.) (serous membrane)
    - (b) productive inflam. with exudation (granulation tissue
    - (c) chronic productive inflam.

Principles of treatment, read Adami Chap. 27, pp. 216-220.

References

Adami. Gen. Path. pp. 375-450

Adami. Inflammation. Ch. I-VII, XV, XVI, XVIII-XXVII. (May omit 8, 9, 10, 11, 12, 13, 14, 17).

or Albutt's System of Medicine, article Inflammation.

Ziegler. Gen. Path. pp. 319-370.

Ewing. Syllabus of lectures on inflammation and associated conditions, 1905.

CARDIO-VASCULAR SYSTEM.

HEART.

Function of the heart.

Hypertrophy—is not primary, is preceded by distension.

- *distension*
- *dilatation*
- *simple hypertrophy*
- *excentric-hypertrophy*

Incompetence—insufficiency

- *relative*—especially likely to happen in right heart.
- *actual*

Distension of right ventricle outcome of (1) incompetence or stenosis of pulmonary valves; (2) obstructive disease of the lungs and pulmonary circulation or (3) obstruction of flow of blood through left heart.

Results of incompetence.

Regurgitation—overloading of chambers into which blood regurgitates—distension of same—followed by compensatory hypertrophy—sooner or later damming of blood on the venous side of the heart.

Incompetence frequently associated with stenosis.

PERICARDIUM.

Pericardial sac normally contains more fluid than other body cavities.

Hydropericardium—due to obstruction of flow of blood in right heart; slow dissolution, incompetence, lung disease and other diseases which lead to distension of the right heart.

Pneumopericardium—puncture or bacteria

Hemorrhages—petechiae, ecchymoses

Pericarditis. Hematogenic or by extension; may be extension from pneumonia, or endocarditis, by traumatic pericarditis (in cattle).

Acute pericarditis

- *fibrinous*, the ordinary form
- *sero-fibrinous*
- *purulent*, usually *fibrino-purulent*.

Chronic productive pericarditis, arises from the acute or insidiously. thickening of membrane by increase of fibrous connective tissue.

May be adhesions between two layers of pericardium, complete or partial *synechia.*
villous heart, especially frequent in dogs.
Tuberculous pericarditis.
Actinomycotic pericarditis.
Traumatic pericarditis common in cattle—swallowing foreign bodies.
Melanosis maculosus in calves.
Tumors, not common. primary are rare; secondary by metastasis and by extension from mediastium.

MYOCARDIUM

anemia—may be partial or general, pericardial exudate or hydropericardium, blocking of coronary vessels.
hyperemia.
hemorrhage, infectious diseases, e. g, anthrax, septicemia hemorrhagica, rinderpest, fowl cholera, morbus maculosus, mercurial poisoning, sometimes agonal; blocking of arteries, etc.
degenerations, from arterial anemia, cloudy swelling, fatty degeneration, fatty infiltration.
necrosis, in small areas, as in livers of cattle, frequent in heart muscle, due to B. Necrosis (Bang)
Calcification and ossification, of right auricle in horses.
Myocarditis.
(1) *acute*, from infection or intoxication
(a) *parenchymatous* or *diffuse*, may produce incompetence by relaxation of muscles.
(b) *fibrino-purulent*, traumatic, the most frequent in cattle.
(e) *purulent*, disseminated by metastasis through blood, e. g. after parturition in cow and bitch, strangles, navel ill and similar wound infections.
(2) *chronic productive* (interstitial), ordinarily accompanies endocarditis, eventually the sequel of the cellular infiltration in parenchymatous myocarditis.
Rupture of heart—muscle weakened by disease.
Hypertrophy—due to
(1) obstruction to outflow of blood
(a) endocardial, stenosis of valvular orifice,
(b) arterial, diminution of lumen, sclerosis, etc.,
(c) pericardial, from complete synechia.

(2) increase in volumne of blood to be propelled
    (a) plethora,
    (b) from regurgitation.
(3) increase in rate of flow of blood
    (a) in tachycardia (in exophthalmic goiter)
    (b) in athletes.

Tumors, mostly secondary.

primary	fibroma	rhabdo myoma	rare
	myxo-fibroma		
	lipoma (man)		

secondary—carcinoma, sarcoma, lympho-sarcoma, osteoid sarcoma, melano-sarcoma.

Animal parasites
Sclerostoma equinum.
Filaria immitis, in dogs.
Echinococcus, in cattle
Cysticercus cellulosus, in swine
C. inermis, in cattle.
larvæ of other nematodes then Filaria

ENDOCARDIUM

valves are formed by reduplication of endocardium with an increased amount of supporting connective tissue.

In aortic and pulmonary values there are no blood vessels.

Hemorrhages.

Thrombosis, with ulceration of endocardium, stenosis of openings; thrombi are apt to form in cavities in dilatation of the heart, in slowing of blood stream, changes in composition of the blood.

Endocarditis. Acute from intoxication or infection.
lesions often ulcerations with vegetations (thrombi), valves more often affected.

Acute. Swelling and coagulation necrosis of affected region, begins on closing surface of valve, greyish appearance there, shedding of endothelium and thrombus formation, leading to warty or granular nodular formation as villous or polypoid outgrowths (vegetations).

part of valve may be destroyed while rest shows fresh inflammation, first a small opaque yellowish patch, surface uneven.

Chronic endocarditis. Very frequent in dogs, occasionally in horses. Fibrous thickening of valves and inner surface in heart. A vascularization of cusps of valves with growth of cicatricial connective tissue, where was earlier an ulcer, the cusp is weakened by the contraction in scar and has increased strain, thickened valves, edges may grow together.

Fatty degeneration.

Fatty infiltration.

Auriculo-ventricular defects occur in all kinds of domesticated animals, about as often on one side of the heart as the other, rarely double sided in same heart. Dog affected with valvular defects following verrucous and contracting endocarditis. Horse frequently has defective semilunar valves, but often without sequlæ if they remain small. In swine and sheep the valvular vegetations are especially voluminous.

Melanosis maculosus in calves, sub-endocardial as well as sub-epicardial.

Tumors of the heart in domesticated animals are mostly in subendo—and sub-epicardial connective tissue.

BLOOD VESSELS.

vascular function, normal.

thrombo-arteritis, *phlebitis*,

verminous arteritis in horses.

suppurative arteritis, *phlebitis* most often through naval infection in calves and foals.

Arterio-sclerosis most frequent in aorta, first greyish semi-translucent patches in intima, may have gelatinous appearance (mucoid degen.), later patches become harder, have more cartilaginous appearance, fibrous connective tissue formed, degenerates becoming a structureless mass, with this may be fatty degen. of cells, and production of granular, shreddy detritus (*atheroma*) resulting in necrosis, often is deposition of calcium salts in this in advanced stages (calcified plaques), may break down into a shallow ulcer; on such ulcers thrombi may be formed.

Experimental arterio-sclerosis, due to strain hypertrophy and overstrain atrophy.

Fatty degeneration

Fatty infiltration

Calcification in arteries in horses, in cattle sometimes.

Aneurisms.

CAPILLARIES.

Thrombosis
aneurisms
degenerations
References:
  Read Krehl 2d. ed., pp. 35-44
  Adami and Nicholls, pp. 115-203
  Kitt S. 334-411.

BLOOD.

The blood as a tissue
  formed elements
    red corpuscles
    leucocytes
    blood plates
  plasma
    water, inorganic salts—phosphates, chlorides, carbonates and sulphates, iron, serum albumin and small quantities of urea, uric acid, glucose, glycogen, fat, acetone, ferments—oxidase, lipase, proteolytic, glycolytic, diastatic.

Knowledge of the changes in chemical composition is of undoubted importance; but substances are present in small amounts and are of complicated structure. Importance of these, for example, specific agglutinating or precipitating substances in certain infectious diseases, e. g. glanders, typhoid, etc., substances producing immunity.

Main reliance in examining blood is on changes in the formed elements and in amount of hemoglobin, changes in red corpuscles, changes in Hb., changes in leucocytes, presence of parasites.

Changes in quantity and quality of blood.
  affecting red corpuscles and hemoglobin.
  plethora—test too complicated for clinical use;
  amount of blood is increased by proper hygienic measures.
  polycythemia increased no. per cmm.
    produced in anhydremia, by withholding water, profuse sweating, watery diarrhea (infectious diseases or by purgative), rapid exudation; in venous stasis, with increased exudation, e. g. cardiac insufficiency, pneumonia, emphysema and thrombosis of lungs, asphyxia, chloroform anesthesia (3 mill. in horses in 1½—2 hrs.).

cold baths, massage, muscular exercise and electricity.

Local polycythemia by ligature, pressure of tumor, partial paralysis, after certain drugs,—pilocarpin, eserin, Glauber's salt, phosphorus.

Age, sex, pregnancy, parturition, high altitude.

Anemia. Tallquist's Hb. scale.

Oligemia

oligocythemia

oligochromemia

Anemia occurs in a variety of conditions, the most important of which are :—after hemorrhage, after marked exudation, in diminished nutrition, in diminished activity of the blood forming organs, in increased destruction of red corpuscles.

Changes in blood in anemia

- obligochromemia
- anisocytosis
  - megalocyte
  - microcyte
- poikilocytosis—poikilocyte
- polychromasia
- punctate bosophilia
- nucleated red cells
  - normoblast
  - megaloblast
  - gigantoblast
  - microblast

Varieties of red corpuscles

- non-nucleated
  - normocytes—small, medium, large
  - microcytes
  - megalocytes
- nucleated
  - normoblasts
  - microblasts
  - megaloblasts
  - gigantoblasts

The blood in normal domesticated animals :

Species	Red corpuscles million per cmm.	leucocytes per cmm.	hemoglobin per cent.
Horse	6½-9	4000-9000	
Average	7.215		94
Cow	4.8-7.9	2300-10,600	45-85
	6.15	6000	60
Sheep			
	8	8000	
Goat	9-10		
	9.976	9200	
Dog	5.6-6.5	6000-10,000	
	6	8200	90
Cat			
	6.6	13,300	
Swine			
	7.9	19,000	88
Rabbit	5-8		
	6	10,000	96
Guinea Pig	4.25-6	5550-21,000	85-100
	5.276	10,000	94

	normal	pathological
varieties of leucocytes	lymphocytes	myelocytes
	large mon.	eos. myel.
	polymorphs	plasma cells.
	eosins	
	mast cells	

Leucocytosis

polynuclear leucocytosis, lymphocytosis, eosinophilia, basophilia

Inflammatory leucocytosis

characterization

occurrence in stranges, pyogenic infections, abscesses, fistulous withers, quittor, suppurations, wound infections, pneumonia (croupous, pleuro—, gangrenous, broncho—), pleuritis, tetanus, muscular rheumatism.

Course observed in patients; best seen in experimental cases.

initial stage (leucopenia)

increase (in from ½ to 4 hours usually)

polymorphs increase 85–95% ; eosins decrease ; eosins reappear at time of crisis.

Measures the relation of severity of infection to resisting powers of individual.

Infection mild, resistance good, small leucocytosis.

Infection less mild, resistance less good, moderate leucocytosis.

Infection severe, resistance good, very marked leucocytosis.

Infection severe, resistance poor, no leucocytosis.

Post hemorrhagic leucocytosis
Ante mortem "
Leucocytosis of digestion
" " new born
" " post parturition and pregnancy
" " exercise and massage
" " cold baths

Experimental leucocytosis after administration of:
quinine, salicylate, mercurials and antimomials,
oil of mustard and vesicants, tuberculin, mallein, thyroid extract, nuclein, antitoxin, lysol injections.

Lymphocytosis in young, after digestion, after administration of thyroid extract, pilocarpin, tuberculin, quinine, after splenectomy, anemia (relative), with most splenic tumors, during the initial stage of inflam. leucocytosis, in infectious diseases with acute hyperplasia of lymph tissue, in *lymphatic leukemia*

Eosinophilia.

high in young in man and dogs.

helminthiasis, Scherostoma equinum, Trichinella spiralis, mange (Sarcoptic, Psoroptic), chronic skin diseases, depends on intensity of dermatitis more than on extent or duration of the disease. See Welch's art. Abstracts of work done in laboratory of Veterinary physiology, No. 7, 1910, p. 17,

Basophilia

Leucopenia

fasting, malnutrition, after short hot baths or long cold baths, during initial stage of inflam. leucocytosis, in anemia (many cases), in tuberculosis (often in non-septic), after injections of ergot, tannic acid, sulphonal, atropin, agaricin, picrotoxin, peptone, diastase.

Leukemia

- characterization
  - (1) *lymphatic*
  - (2) *mixed-celled* (myelogenic)
- Changes in the blood; red corpuscles diminished, may be erythroblasts present.
  - *Lymphatic*
    - Leucocytes much increased 100,000-1,000,000 per cmm., lymphocytes 92–99%, other varieties scarce.
  - *Mixed-celled* (myelogenic)
    - leucocytes much increased 100,000-1,000,000, finely granlar myelocytes, polymorphs, small lymphocytes, large lymphocytes, eosins, eosinophilic myelocytes, mast cells
- Pathological anatomy
  - Bone marrow—red marrow of ribs, vertebra, etc., lighter colored and of firmer consistency, cellular hyperplasia of lymphocytes or myelocytes, extension of lymphoid marrow in shafts of long bones and an atrophy of fat marrow. Spleen enlarged, rich in cells, sometimes pulp cords, sometimes follicles or both, usually distinction not so clear as in normal, sometimes boundaries indistinguishable, stroma may be much increased.
  - Liver usually enlarged, capillaries widened and contain many leucocytes (lymphocytes), cellular infiltration between lobules, may be collection of cells in the lobules.
  - Lymph glands enlarged, cellular hyperplasia, boundaries of follicles and medullary cords are commonly apparent though sometimes indistinct, lymph sinuses and blood vessels contain many leucocytes, reticulum sometimes thickened making gland more firm.
  - May be masses of lymphocytes (in lymphatic variety) in other organs, kidney, skin, serous membranes, central nervous system, etc., appearing as grayish nodules.

Pseudo-leukemia group.

- Marked enlargement of lymph glands and usually of the spleen.
- Group 1. hyperplasia of lymph cells. Lymphocytomatosis.
- Group 2. chronic productive inflam. changes, increased conn. tissue and endothelial cells; occurs in tuberculosis, syphilis, and in cases with cause unknown.
- Relation of leukemia to pseudo-leukemia and lympho-sarcoma. See Report of Director of N. Y. State Veterinary College for 1910, p. 130.

Pernicious anemia.

References.

Adami and Nicholls, pp. 19-32, 83-102.
Burnett, Clinical Path. of Blood of Domestic Animals.
Ewing, Clin. Path. of Blood.
Kitt, pp. 412-459.

SPLEEN

Shape in different animals.

Physiological variations in size.

Hyperemia—*active* from intoxication or infection.

passive enlarged firmer, dark red color, capsule tense, opaque; often extensive pigmentation from disintegration of red corpuscles.

Infarction from emboli, generally first white, later may become hemorrhagic.

Acute hyperplastic splenitis.

enlarged in infectious diseases to ⅓ to 2-6x, capsule distended, tissue at first firm and dark red (hyperemic), becomes soft or even diffluent, color paler; extensive hemorrhagic extravasations not uncommon.

Microscopically overdistension of vessels, proliferation of cells both in pulp, or in Malphigian bodies, or in both; degeneration of cells—fragmentation of nuclei, lack of nuclear staining, cellular detritus, fatty degeneration; usually terminates in resolution. May proceed to rupture, to suppuration or fibrous hyperplasia.

Suppurative splenitis. In septic processes. Usually embolic, abscesses usually multiple, or by extension from adjacent suppuration.

Chronic indurative splenitis.

diffuse hyperplasia of conn. tissue, from repeated attacks of acute splenitis or from long continued venous stasis (cirrhosis of liver).

Capsule thickened, consistency of organ usually increased, associated with parenchymatous changes. May be adhesions, great increase in trabecular tissue.

Tumors.

Sarcoma sometimes primary, Melanosarcoma usually secondary, lymphosarcoma. Carcinoma (metastatic).

LYMPH GLANDS

Structure—cortical nodes, medulla, trabeculæ and capsule, lymph sinuses.

Function—filter for lymph, on guard at all orifices and channels of body, to form leucocytes (Var. I).

- Lymphatic tissue more prominent in young, becomes less in old.
- Hyperemia practically only inflammatory. Normally contain little blood. Glands reddened, enlarged, juicy.
- Hemorrhages, in inflammations, occur readily.
- Edema inflammatory or part of general anasarca.
- Pigmentation.
- Simple atrophy in old age.
- Amyloid and hyaline degeneration.
- Calcification—common in tuberculosis of cattle.

Lymphadenitis, due to infection somewhere, a common condition (one of the most common).

Acute lymphadenitis—glands swollen, injected and soft, on section, surface smooth, reddish grey, in severe cases studded with yellowish grey foci (areas of necrosis).

Microscopically—normal structure often unrecognizable, blood vessels distended, hemorrhagic extravasations; lymph sinuses filled with desquamated endothelial cells, red corpuscles and leucocytes; round cell proliferation in nodes, degeneration of lymph cells, nuclei disintegrated, cells swollen. Ends in severe cases in necrosis, may end in suppuration, or in induration where irritation is not severe but long continued.

Suppurative lymph-adenitis—from pyogenic infection, gland softens, suppuration.

Chronic indurative lymph-adenitis—from acute or may start insidiously. Gland enlarged, firm and dense; hyperplasia of conn. tissue of capsule, trabeculæ and may also be of recticulum, decrease of lymph cells from encroachment of conn. tissue.

Pseudoleukemia group. Hodgkin's disease. Enlarged lymph glands without increase of leucocytes in circulating blood.

Sub-group I. enlarged glands chronic productive in flammatory process.

Sub-group II. Enlarged glands are growths of lymphocytes with more or less framework of conn. tissue. Aleukemic lymphocytoma. Hodgkin's disease.

Tumors—lympho-sarcoma, sarcoma, fibro-sarcoma, enchondroma and carcinoma (metastatic).

RESPIRATORY SYSTEM.

Primary function
  Exchange of O_2 and CO_2
  internal respiration, the more important takes place in all the tissues. Some of symptoms of anemia are like those of interference with breathing.
  external respiration
    (1) disturbances in mechanism by which air reaches lungs and and effects of these,
    (2) disturbances in the air sacs and their effects,
    (3) changes in composition of air entering lungs.

I. The respiratory mechanism.

(a) air passages. They are long, so air is warmed and made moist before entering the air cells and is freed from dust and foreign particles and so is sterile.

Complete obstruction leads to death in a few minutes; incomplete to inspiratory or expiratory dyspnea or both, followed according to amount by death by slow suffocation, by asphyxia or by adaptation of respiration and needs of body to the lessened supply of oxygen. With obstruction to inspiration, respiration becomes slow and labored, diaphragmatic contractions deeper and other accessory respir. muscles into action to make thorax as large as possible. The expirations are short and labored. Inspiratory stridor. The reverse occurs in expiratory obstruction. Expiration slow and labored; the accessory muscles called into action are those which most lessen the size of the thoracic cavity, especially the abdominal muscles and those bringing the ribs closer together. With one bronchus affected a period of rapid respiration followed by indications of adaptation, the one lung being able to perform the function of both. Rapid period due probably to different periods at which vagus inspiratory and expiratory stimuli are started in the two lungs.

(b) *The muscles of respiration.*—In ordinary quiet breathing inspiration is active, expiration passive.

(c) *The pleural cavities* bring about uniform expansion. When lungs are attached to pleura, as in adhesions, there would be unequal expansion. There is negative pressure in pleural cavities.

Pneumothorax—deeper respiration
  open, pressure positive, lung collapses.
  closed, some slowing of rate.

Pleural effusions—lessen space that may be occupied by lung, may bring about complete collapse. With any quantity of fluid, elasticity of lung is not effective. Fluid is under positive pressure till amount becomes very small.

(d) *The nervous mechanism*.—Several separate factors, several muscles, which must be regulated by center. Pulmonary branches of vagus the main afferent nerves of respiration. Center stimulated by increased carbon dioxide and depressed not by increased oxygen but by decreased carbon dioxide tension. Over ventilation of the lungs may bring about arrest of breathing due to lack of carbon dioxide, e. g. in surgical "shock," painful injuries, to revive do not need more air, but less or rather more carbon dioxide.

Coughing.—Reflex due to irritation in the larynx (usuully), in the lungs, or of the pleural surfaces. Deep inspiration followed by closure of glottis during the first part; with sudden opening of glottis, air under pressure is freed carrying with it mucus, etc., in bronchi, trachea, or larynx.

Dyspnea is manifested under the following conditions:

(1) conditions of severe hindrance to entrance of normal amount of air (a) in the air passages (leading to diminished ingress and egress of air (foreign bodies, imflammatory or other narrowings of larynx, trachea or bronchi or compression from without; (b) in the lungs themselves—collapse, diminution of ventilating surface by exudates into the alveoli, tuberculous and other growths in lung substance, destruction of lung substance (gangrene, cavitation, etc.), fibroid inflammation, atrophy, emphysema.

(2) conditions affecting the muscular mechanism of respiration (a) diseased conditions, inflam. etc., of the diaphragm or other respiratory muscles; (b) pathological condition of the main and secondary centers and of the nerves forming the respiratory arc, paralysis or irritation of the centers, trauma or destruction of the afferent nerves (vagus) or again of the efferent nerves (more particularly of the phrenics.)

(3) conditions affecting the circulation of the blood; (a) through the lungs (obstructive heart disease emphysema); (b) through the medulla.

(4) conditions modifying the constitution of the inspired air—rarefaction of the air at high altitudes, reduction

of the amount of contained oxygen, increased carbon dioxide content, presence of carbon monoxide which unites with hemoglobin preventing the taking up of oxygen.

Asphyxia. Three stages.

1st. of increased amplitude as well as rate of the respiratory movements.

2nd. with increase of the expiratory movement out of all proportion to the inspiratory, the expiration becoming prolonged, the inspiration short and convulsive. Whole musculature of the body may be called into action to produce these violent expirations.

3rd. violent expirations cease almost suddenly and slow, deep inspirations manifest themselves; mouth widely open, head back to give most free entrance of air into trachea, body stretched, complete insensibility. Inspiratory movements further and further apart, weaker and weaker; in few minutes last gasp. These changes accompanied by changes in circulation most marked a rise of blood pressure, in third stage heart begins to fail, poisoned by venous blood.

II. Disturbances in the air sacs and their effects.

(a) those in which more particularly the entrance into and egress from the air sacs of the proper amount of air is prevented.

by want of distension, as in atelectasis and collapse;

by collection within them of fluid or solid matter,

(1) by serous fluid as in cases of acute or chronic congestion,

(2) by blood as in rupture of an aneurism in branch of pulmonary artery or of aorta into trachea,

(3) by water or other fluid from without,

(4) by inflam. exudates and migrated cells, as in pneumonia. Effects depend on extent of lung tissue involved and on the cause. May not be noticeable, may be rapid respiration, dyspnea and finally adaptation or asphyxia and death.

Diffusion of products of coagulation and of death of red corpuscles in cases of hemorrhage, of toxins and cytolytic products in cases of pneumonia sets up febrile state, and among other things affects the respiratory centers, inducing an increased rate of breathing out of proportion to the extent of tissue involved.

In a lobar pneumonia the pleura is involved. The pleural surfaces painful—inhibits thoracic movements—another cause of rapid and shallow breathing. May fill air sacs of entire lung or lower parts of both without fatal result.

Fluid agents tend to accumulate to lower portion—vary with position of patient and may set up disturbances in respiration during change.

Greater interference with circulation with distention of air sacs by solid contents. In collapse and atelectasis no air pressure in air cells, blood flows unhindered, vessels become dilated.

When serous fluid in sacs, in active or passive congestion. When passive not in lung but obstruction in left heart, mitral or other disease. Dilation of capillaries which continues, however great the accumulation of serous fluid.

With coagulation, pneumonia and hemorrhagic infarcts, passage of more and more substance into air sacs, these become over-distended and pass from state of congestion to narrowing and compression of capillaries between air cells. If much lung affected increases work of right heart, dilatation of right heart, and some incompetence. Local necrosis and gangrene are comparatively rare as a result of the anemia. Do show sometimes.

(b) changes in walls of air sacs hindering aeration.
atropic—in vesicular emphysema.
interstitial deposits.

Emphysema overdistension

(1) increased difficulty of expiration leading to raised intraalveolar pressure.

(2) diminished elasticity of walls.

Bronchiectasis. dilatation, result is diminished aeration of blood.

Interstitial deposits. fibrous conn. tissue as in chronic interstitial pneumonia, actinomycosis or neoplastic; leads to compression and diminution or even occlusion of air spaces. Also obstructs circulation—double effect.

MORBID ANATOMY OF THE LUNGS

Blood supply—two sources.

Anemia

- *local*—hindrance to flow of blood to lungs, as pressure of tumors, emphysema, etc.
- *general*—general oligemia, color *clear*, *yellowish rose tint*, in high grade may be *white yellowish tint*. That is the case with emphysema.

Edema.

Of common occurrence. The most frequent cause is relaxation at time of death. May be of entire lung or one or more lobes. Lungs become heavier and firmer than normal, pit on pressure, on section tissue filled with thin, watery fluid, shown easily by pressing lungs with hands. When fluid removed lung regains normal crepitant state.

Hyperemia.

- *passive*—with cardiac lesions stenosis, insufficiency, most with mitral insufficiency ; in chronic cases there is an increase of fibrous tissue in walls of lung (*cyanotic induration*) in *brown induration* is also hemorrhage by dispedesis, blood pigment freed and taken by large clear mononulcear cells ("heart failure cells")
- *active*—functional, over exercise
  - lessening of air by obstruction or narrowing of upper air passages
  - irritating gases
  - collateral
  - inflammatory
- *hypostatic*—with obstruction to free outflow of blood from the lung or lack of power in right heart, lower part affected. In some infectious diseases, fatty heart; cloudy swelling of heart.

Hemorrhage.

Hemorrhagic spots in suffocation or asphyxia are dark red punctiform sharply circumscribed hemorrhages, may be size of small coin, on sub pleura over lungs, are numerous on costal pleura, especially at greater angle of ribs. Occur in cases of death with appearance of high grade difficulty of breathing. Called by Kitt "haemorrhagia per suffocationem sive asphyxiam",

Hemorrhagic infarcts.

Embolism and thrombosis, fat, air, tumors,

atelectasis
congenital
obstructive—bronchial obstruction
compressive, external pressure by fluids, air, elevated diaphragm, aneurisms, tumors, etc.

emphysema
interstitial, interlobular and bullous.
vesicular or alveolar—simple, ectatic, atropic.

An acute vesicular form of emphysema occurs in last stages of all diseases where death occurs with marked difficulty of breathing.

Chronic form is frequent in horses. The entire lung enlarged.

Effect of emphysema. In beginning enlargement of alveoli may assist in exchange of gases. But as they become more enlarged, the walls press on the blood vessels and hinder flow of blood, so hinders respiratory function. Blood cannot enter lung so well, is held back in right heart—enlargement of rt. heart. Passive hyperemia there and in venous system.

Inflammation of lungs from three sources—through blood, lymph, bronchioles

In inflammation in lungs find exudate in alveoli as a rule instead of in walls which are thin and permeable.

croupous, *fibrinous*, lobar pneumonia, e. g. principal appearance in contagious pleuropneumonia of horses, contagious pleuropneumonia of cattle, hemorrhagic septicemia of various animals, often in swine plague, occurs in glanders in carnivora (cats, lions)

Is due to germs (specific?)

Characterized by exudate, fibrinous in alveoli, affects alveoli and bronchioles; usually unilateral.

several stages.

1. *congestion*—engorgement of blood vessels, alveoli may contain some serum, a few corpuscles and few leucocytes.
2. *stage of red hepatization*—lung dense, tumefied (larger than normal) of consistency of liver, alveoli filled with fibrinous exudate, more or less red corpuscles and leucocytes. Leucocytes in walls of alveoli. Reaches its height at 5-6 days. Pieces sink in water.
3. *stage of grey hepatization*. Affected lobes dirty greyish in color, alveoli filled with leucocytes and fibrin. May scrape off graular material; pleura with fibrinous exudate.
4. *stage of resolution*. Fibrin granular, absorbed. Alveoli filled with leucocytes; on scraping surface of section con-

tents of alveoli come out as plugs. Resembles pus. Fatty degeneration of leucocytes. Exudate absorbed or partly expectorated. Lymph glands swollen (softer than normal). May be gangrenous areas, small usually, may be large in affected part. Heart often fatty, also liver and kidneys.

Catarrhal or broncho-pneumonia.

Cause—irritants as dust, "cold", in other diseases, often terminal, hypostatic. Staphylococci and streptococci. Most frequent in dogs (Kitt), in sheep and goats somewhat common—due to lung worms. Is lobular, extends to alveoli from bronchioles. First bronchitis, peribronchitis, then pneumonia.

Differentiation.

Catarrhal begins with bronchitis, has irregular course, ends by atelectiasis (in young pigs) and induration, is lobular—limited area.

Gangrene of the lungs.

Most often in horses following hemorrhagic croupous pneumonia, in cattle following introduction of foreign bodies from rumen, and as metastatic (embolic) may be one area or several. More rarely gangrenous pneumonia occurs in dogs.

Suppurative pneumonia.

Abscesses.

PLEURÆ.

- Hydrothorax
- Hematothorax
- Emphysema of mediastinum
- Pleuritis
  - *serous and serofibrinous*
  - *hemorrhagic*
  - *purulent, suppurative*
    - empyema of the thorax
  - *chronic productive*

Tumors

Parasites

RESPIRATORY PASSAGES.

Stenosis of trachea

Bronchiectasis and bronchi stenosis

Hyperemia

Hemorrhages tracheo
broncho

edema of the glottis
laryngitis, *tracheitis*, *bronchitis*,
    croupous, diphtheritic, necrotic
    catarrhal
        serous, desquamative, purulent
    chronic prodvctive
    suppurative
tumors.

References.

Adami and Nicholls, p. 237-338.
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DISEASES OF THE STOMACH AND INTESTINES.

STOMACH.

Function
Blood supply, affected by disease of liver, directly and indirectly.
Volvulus in dogs
Bezoars
Dilation of stomach in dogs
Rupture
Autodigestion of mucosa—post mortem maceration,
Hyperemia—functional, active, passive
Anemia
Hemorrhage

Acute catarrhal gasrtitis.

morbid changes may be so slight as to pass unnoticed without anamnesis.

Mucosa of pyloric part of stomach shows swelling with redness, congestion in patches or diffuse. Small hemorrhages, punctiform or larger, give mucosa a dotted or spotted appearance. Mucus incresed glairy or sometimes purulent, may be blood stained.

Microscopically, secreting cells show cloudy swelling, goblet cells numerous (mucous degeneration), cellular infiltration (round cells ?) of conn. tissue between glands and in the submucosa. Interstitial blood vessels hyperemic, swelling and desquamation of endothelium of lymph vessels; on surface stringy mucus containing clumps of epithelial cells, red corpuscles and leucocytes.

Chronic catarrhal gastritis.

Mucosa covered by greyish, thick, tenacious mucus, may be more purulent, sometimes streaked with blood, more or less thickened, forming ridges or folds, color dark brownish red, in advanced cases slate grey. Discoloration due to blood pigment from hemorrhage. Mucosa may be increased to x 5 normal thickness. Rarely mucosa thinner than normal. Mucosa may have warty appearance due to proliferation of fibrous conn. tissue between the glands. May be outgrowth of glands giving an adenomatous appearance. These two sometimes called *hypertropic gastritis*. Other coats may show hyperplasia, so stomach wall may be much thicker. Sometimes stomach, dilated by relaxation of muscles, is thinner.

Mic. most striking feature is cellular infiltration in interstitial areolar tissue, especially in outer layers of mucosa, leading to more or less separation of glands. Ducts are catarrhal, often tortuous, dilated, cystic, secreting cells enlarged and more numerous.

Other forms of gastritis

hemorrhagic leading to erosions in mucosa,

phlegmonous,

tuberculosis occurs, but is rare.

poisoning

Gastrophilus "erosions" shallow not of importance in ordinary cases

Parasites, long list. (See text books).

Tumors.

INTESTINES.

Hernia

strangulation (by lipoma)

volvulus

prolapse

invagination

intusseption, happens sometimes at death.

dilatation (sometimes is distended by gas after death).

phytoconcrementa, pili concrementa, bezoars, hair, wool, bristles, sabbura coli, mixture of sand, silt, hay and straw and other portions of fodder, enteroliths

hyperemia

hemorrhage diffuse, capillary, punctiform, hemorrhagic erosions, diffuse stasis.

necrosis

gangrene

Inflammation

duodenitis
jejunitis
ileitis
typhlitis
coliti
proctitis
gastro-enteritis

Acute catrrrhal enteritis.

one of the most frequent affections. May accompany many diseases. Mucosa appears in places or throughout its entire extent reddened, (hyperemia), sometimes confined to villi, sometimes about Peyer's patches which are swollen and project as nodules "follicular catarrh". May lead to small crateriform ulcers. Increased amount of secretion, mucus may be blood stained. Submucosa infiltrated with fluid. Intestinal contents in large intestines thin.

Chronic catarrhal enteritis.

Mucosa dark red in streaks and spots, sometimes slate grey, vessels dilated and tortuous, surface covered by glairy mucus. Follicles are at first swollen, later atrophy and disappear when mucosa has an areolated appearance, contents watery. Mucosa generally thickened, may be thinned, compact and wrinkled by proliferation of interglandular and submucous conn. tissue.

In large intestine follicles frequently prominent as nodular swellings, may form ulcers.

Hemorrhagic enteritis, colitis, etc.

diphtheritic, superficial necrosis.

croupous or membranous

ulcerative hog cholera, i. e. B, necrosis, tuberculosis

Tuberculosis

Actinomycosis (rare)

Glanders

Parasites, very many kinds and many individuals.

See Law, Kitt, Neumann.

Tumors

Lipoma in serosa containing adipose tissue, in mesentery, omentum, diaphragm, sarcoma, lymphosarcoma, melanoma, leiomyoma, adenoma, adenocarcinoma, carcinoma,

Sequelæ of diseases of the intestines.

Many complications, in general all diseases of the intestines tend to interrupt the digestive function, destroying absorption, hindering excretion, shown both as diarrhea and constipation.

Law of vicarious function. When one organ or system fails to perform its function, others will attempt to take up the work and do it as well as they can, e. g. elimination of urea from skin in chronic Bright's disease, stomatitis and diarrhea not an uncommon result; ulcer of the lower bowel sometimes. Exposure to heat and cold frequently followed by diarrhea.

Number of serious disturbances may arise from resorption of secretions. A certain amount is normal. Water in intestines, feces more solid in large than in small. A considerable amount of bile salts and pigments are resorbed from the intestines. Under ordinary circumstances resorption is followed by no ill effects, when excessive there is.

Bile is absorbed into the circulation when there is obstruction to its flow into the intestines. By calculi, catarrh of fine ducts, tumors of bile ducts or head of pancreas, enlarged glands or abscesses at hilus of liver and tumors of liver. Jaundice (icterus), digestive processes interfered with, feces clay colored, pulse slowed dullness, itchiness of skin, lessened coagulability of the blood. Resorption of bile depends on degree of obstruction and viscidity of bile. With thicker bile less obstruction produces jaundice. Thick viscid bile may be absorbed in cases of catarrh of the finer bile ducts.

Obstruction to free outflow of pancreatic juice may be followed by serious results. May lead to fibrosis, if of a lateral branch—cyst formation—may be very large cysts. If large areas of secreting cells are destroyed may be interference with the internal secretion—glycosuria, obsruction may be by tumors in head of pancreas or calculi. If it allows bile to enter pancreas ; hem. pancreatitis follows with fat necrosis. Fat necrosis occurs in other conditions of disease of the pancreas, rarely in cases of suppuration.

(Read in Adami and Nicholls, pp. 372-380.)

PERITONEUM, OMENTUM AND MESENTERY.

Hydrocoecle,
hematocoele,
pyocoele,
pneumatosis peritonii,
emphysema of the intestines, peritoneum,
  some cases due to B. coli, gas cysts always on small intestine; gas cysts come from lymph vessels, often in healthy slaughtered swine.
ascites.
serous peritonitis
chronic productive peritonttis, may be adhesious between intestine and body wall, intestine and sometimes 'liver, different loops or part.
Omentitis, mesenteritis, perihepatitis, perisplenitis,
perimetritis, perigastritis perityphlitis.
tuberculosis
actinomycosis
Parasites.
  Filaria, Sclerostoma bidentatum,
  Stephanurus denticulus, Lingulatula denticulata,
  Cyysticercus tenuicollis, Echinococcus.
Tumors
  Carcinoma, primary once reported in horse ?
  melanoma, sarcoma, lipoma.

LIVER.

Appearance of liver in cadavers.

	color cold	normal weight
horse	chocolate brown	3-4 Kg. in old; 6-8 in younger during digestion.
cattle	chocolate brown (in steer and oxen, red brown)	4.5 Kg. average
sheep	clear chocolate brown.	370-870 gm.
goat		370-870 gm.
swine	clear chocolate brown	1-2½ Kg. (in fasting fattened swine 500 gm. difference).
dog	bright red brown	small 90-250 gm.; large 500 gm.—1½ Kg.
cat		80-120 gm.

still with body heat livers are clear brown to greyish yellow.

Hemocysts serosa hepatis. In sound slaughtered calves peculiar large cysts, congenital blood cysts in the liver. Besides these cysts liver otherwise normal.

Anemia. Part of a general anemia or due to local disturbance as pressure on the liver or swelling of parenchyma.

Hyperemia.

Active. Physiological after a meal. Patholog, in early stages of acute hepatitis and in all cases of congestion of the gastro-intestinal vascular system.

Passive. Brought about by anything that raises the pressure in the hepatic vein and vena cava. Among others stenosis or insufficiency of mitral or tricuspid valves, cardiac weakness, emphysema of lungs, indurative pneumonia, right sided pleural effusions, aneurisms, tumors, or enlarged glands pressing on the post cava.

In early stages liver is enlarged, soft, full of blood, which drips out on section. Color dark, purplish red; after blood has been drained out, liver will be found to be somewhat shrunken (*red atrophy*). Later liver is smaller, firm, with finely granular surface. On section, appearance like that of a nutmeg (nutmeg liver) due to central congested part of lobule being depressed below general surface, the periphery being pale yellow or yellowish brown and swollen, due to fat in secreting cells. May be redder patches—regenerated lobules. In most advanced stage liver small and hard, due to conn. tissue formation (cyanotic induration).

Hemorrhage, rupture, infarction (rare), acute yellow atrophy, infections,

Cloudy swelling.

Fatty liver, common sequel of cloudy swelling, occurs in severe anemias, acute yellow atrophy, congestion, intoxication (phosphorus and arsenic).

Amyloid, occurs in horses, dogs, cattle, sheep, rabbits and poultry. In cachectic conditions, suppuration.

Liver generally enlarged, rounded edges, consistency increased somewhat, amyloid centers sometimes evenly spread, sometimes irregularly. Liver contains but little blood. In hens are scattered nodules. Gross appearance gelatinous, semi-translucent. Liver cells atrophied and fatty.

reaction	Lugol's sol.	mahogany red
	H_2SO_4 dilute	grey to violet blue
	methyl violet	aniline red.

Necrosis. As multiple nodules, often in livers of cattle and sheep and pigs due to B. necrophorus. Liver enlarged, foci from size of pea to walnut, round, hard, dry, yellowish and often surrounded by capsule of conn. tissue.

Chalky nodules in horse livers. May be glanders, more likely to be parasites.

In sheep Oesophagostoma columbianum.

Atrophy. In dog and cat can lose ⅔ of volume in starvation.

Compression atrophy. Of some part of liver not infrequent; most common is the atrophy of right lobe in old horses, sometimes also the left, from pressure of the colon.

Acute parenchymatous hepatitis.

Causes—damaged fodder, mechanical, excessive heat, poison, infectious diseases.

Appearance very closely resembling that of cloudy swelling. Liver slightly enlarged, edges blunt, soft and friable; pale yellow, clayey color; lobes may be adherent, perihepatitis, may be adherent to diaphragm.

Mic. appearance; cells swollen and granular, nuclei pale; fatty degeneratiion, hyperemia interlobular; may be collections of leucocytes in portal sheaths.

May be considerable necrosis, pigment in cells, liver becoming smaller. *Acute yellow atrophy* (most frequently seen in lupinosis in sheep and horses) a very acute form of parenchymatous hepatitis *Diminution in size of organ;* softening and yellowish discoloration of parenchyma, liver cells show granular and fatty degeneration, contain pigment, necrosis extensive.

extensive icterus *acute yellow atrophy*, considerable hyperemia *acute red atrophy* (commonly fatal termination).

Suppurative hepatitis.

(1) from infection of bowels or metastasis from hepatic arteries, e. g. in pyemia, glanders and acute infections.
(2) from thrombosis of portal or umbilical veins e. g. omphalophlebitis.
(3) foreign particles from stomach, bowels or blood—fodder, barley awns, parasites.
(4) mechanical injuries, from inflam. of bowels, gall stones, etc.; sometimes minute multiple abscesses, sometimes larger; may be few or many, sometimes confluent, sometimes single.

Chronic productive hepatitis.

Hyperplasia of conn. tissue which in time becomes sufficient to interfere much with function of liver. Due to irritation of toxic substance reaching liver through portal circulation (hepatic artery?) or bile passages. In horses, swamp fever or infectious anemia of horses, in dogs with valvular disease of heart. More than one cause. Cause is ordinarily not severe but is mild and long continued e. g. alkali in swill for pigs. Surface granular, nodular or even lobulated in much protracted cases, capsule thickened, color at first brownish, later light yellow, organ bloodless.

On section shows whitish narrow bands of conn. tissue surrounding the yellowish lobules.

Microscopically first infiltration of small cells in periportal tissue, later development of conn. tissue.

Usually is an interstitial productive hepatitis. Sometimes diffuse, e. g. "Pictou disease" in cattle. Also in other cases besides "Pictou disease." In diffuse form proliferation of conn. tissue within lobules isolating small groups of liver cells. Changes in other organs, hyperemia and chronic catarrh of stomach and intestines, enlarged spleen, ascites, anasarca, hemrhages in various organs.

Parasites.

Coccidosis in rabbits. Distoma hepaticum.

Cysticercus tenuicollis, C. pisiformis, Echinococcus, more often in cattle and swine, only once reported in liver of ass and horse.

Regeneration, in experimental cases can lose ¼, ½ or even ¾ of liver and loss made good by regeneration of remainder.

Tumors. Adenoma in cow, Adeno carcinoma, and carcinoma not so often as in man, sarcoma, (primary is rare), lipoma, telangiescasis, hemangioma.

(melanosis maculosa of calves, spots on surface of liver and in section).

Effects of disease of the liver on other organs.

References.

Adami and Nicholls.
Kitt, pp. 612-708.
Law, Vol. V, pp. 332-364.
Neumann, pp. 421-464.

URINARY SYSTEM.

Structure of the kidneys.

Function of the kidneys.

external excretion seems to be the function, are chief eliminative organs of the body. Other eliminative organs for toxic substances are intestines, liver, lungs and skin. Most of water in urine passes out through glomeruli, discharge of urine influenced largely by circulatory conditions. Blood pressure reduced to 40 mm. Hg or lower secretion ceases ; with increase of amount of blood passing through kidneys the amount of urine is increased. But with renal vein ligated blood pressure in kidney is very much increased but no urine is secreted.

Water of urine is mostly discharged from glomeruli, nature of selective discharge, not a mere filtration, also some simple salts, e. g. peptone, glucose.

In tubules may be some absorption but resorption is secondary, main function of this part is to excrete urea, uric acids and extractive bodies of the nature of toxins. Formation of hippuric acid.

Nerve supply. Form a fine plexus around renal artery, largely vasomotor ; network of filaments in cortex and medulla with ends on Bowman's capsule and in membrana propria of convoluted tubules, probably secretory in function.

Amount of urine seems to depend on glomerular blood supply which in turn depends on the tone of the interlobular arteries. Glomeruli themselves probably passive, no nerves have been demonstrated in them. Constituents may exert some influence indirectly on walls of glomeruli.

Contraction and expansion of arterioles may be due to central nervous influence or directly by action of substances diffusing out from the circulating blood.

Relationship of vascular disturbances and chronic interstitial nephritis.

Continued rise in blood pressure—muscular coat of cortical arteries hypertrophied—degeneration of glomeruli—others undergo compensatory hypertrophy and become more pervious than usual—more fluid passes through.

When glomeruli atrophy or become impervious it cuts off supply from tubules in cortex, especially convoluted, they undergo degeneration. No urine from glomeruli or from the tubules. In both cases the trouble is with blood supply.

Conclusions.

Quantity of urine discharged depends on quantity of blood passing through the glomeruli. Flow depends on difference in pressure between renal artery and vein. Greater arterial pressure without increase in venous then greater amount of blood passing through glomeruli and greater urine excreted.

Pressure in vein increased without increase in artery then urine diminished.

Polyuria.

Brought about (1) by increase in the general arterial pressure without renal change (2) dilatation of cortical arteries without of necessity any rise in general blood pressure (diabetes insipidus). Perhaps another group.

Two groups of diuretics (1) those inducing increased blood pressure with better circulation through the kidneys e. g. digitalis. (2) those having little or no effect on general blood pressure, e. g. dextrose, urea, sodium chlorid and other saline diuretics. Question whether they dilate renal vessels or stimulate the renal cells.

Oliguria and Anuria.

Brought about (1) by direct contraction of afferent arteries (as through action of adrenalin), (2) by lowering arterial pressure, (3) by rise in venous pressure e. g. valvular disease) may be a fourth, obstruction to outflow if it affects both kidneys (either in tubules or in ureters, etc.)

Hydronephrosis.

Hemoglobinuria.

Chyluria.

Aplasia. One may be absent

Cysts. Common. Supposed to be due to retention due to imperfect fusion of collecting tubules with secreting portion.

Oliguria. General
local (anemic infact.)

Hyperemia.

Active, in acute inflam., various intoxications, forcibly acting left heart, and so on.

Passive, usually due to obstruction in general circulation as valvular heart disease, or pulmonary trouble.

Kidney enlarged, firmer than normal, capsule peels off easily.

Stellate veins are injected, surface has dark purple red, or cyanotic appearance. On section organ very firm, drips blood and has uniform dark red appearance.

Microscopically vessels all enlarged and congested. Bowman's capsule contains albumen and may be red cells and tubules containing a few hyaline casts. Some of epithelial cells (those of descending loops especially) contain pigment (from blood). In long standing cases capillary and venous walls are thickened. There is increase in interstitial conn. tissue and sometimes a round celled infiltration in interstitial tissue—"cyanotic induration." In some cases epithelium of tubules is fatty.

Infarction. Usually anemic, due to plugging of one of branches of renal artery.

Arteriosclerotic contracted kidney.

A form of granular kidney due to narrowing and finally obstruction of afferent blood vessels. May be restricted to renal artery and its branches or may be part of a general arteriosclerotic process. Chronic proliferation of intima or media leads to collapse of glomerular capillaries and atrophy of glomerular tufts. Tufts become rounded hyaline masses, glomeruli atrophy. Tubules become collapsed and atrophied. As the various structures atrophy they are replaced by conn. tissue which shrinks forming scar. Surface of kidney becomes granular. Capsule somewhat adherent. Color bright red or greyish red, or may be streaked with yellow, due to fatty degeneration. Organ smaller in size and firmer than normal. Cortex narrowed and distorted. Glomeruli appear as whitish dots. The smaller arterioles are easily recognisable, due to the increased thickness of their walls. Pelvis relatively large and filled with fatty tissue. *Narrowing* of cortex and medulla. May not be easy to distinguish from inflammatory contracted kidney. Cause different, though.

Senile atrophy.

parenchymatous degeneration. Sometimes difficult to draw the line between this and inflammation.

amyloid, not so common in domesticated animals as in man; mostly after chronic suppurative process, not after every suppurative process so must be some other condition. In walls of arteries and arterioles. After tuberculosis in birds, in man after tuberculosis.

Nephritis. Besides kidneys, organs that eliminate toxins are intestines, liver, lungs and skin. Troubles of these organs interfering with their functions add to work of kidney e. g. water from kidney during cold weather, sweat lessened; urea excreted from the skin when kidneys fail to perform their function. Infection most important cause of acute nephritis. Sometimes follows gastroenteritis. May occur after sudden chilling of body. With certain drugs Hg, Pb, cantharides, $KClO_3$, salicylic acid, phosphorus. In course of acute infectious diseases, hog cholera, pneumonia, etc.

Acute parenchymatous nephritis.

Characterized by marked degeneration of epithelium of secreting tubules with congestion and exudation of serum and desquamation of epithelial cells. Degeneration is cloudy swelling and fatty degeneration. In early stage may be difficult to determine whether it is cloudy swelling or actual nephritis. With congestion, exudation and desquamation inflam. shown. Changes in urine. Kidneys usually more or less swollen and edematous. Capsule peels off easily, bulging of kidney through cut. Surface of kidney pale, veins injected; cortex pale, swollen, cloudy, in contrast to the dark red, or bluish red medulla. Sometimes cortex somewhat congested. Oftentimes can see petechiae on surface and in cortex.

Microscopically. Convoluted tubules most affected. Cells swollen, granular, often vascuolated, nuclei stain poorly or not at all. Hyaline or granular casts in tubules, especially in collecting. Desquamated cells often found. Sometimes blood in tubules. Glomeruli show signs of degeneration. Epithelium lining Bowman's capsule swollen and often desquamated. Hemorrhage in capsule sometimes. Sometimes blood and blood casts may appear in the urine. This may be called *Acute hemorrhagic nephritis*. Interstitial tissue swollen and edematous and sometimes a round celled infiltration between tubules, and may be some fibrin between tubules. In some cases glomeruli affected. It is then an *Acute glomerulo-nephritis*. Kidney doesn't show much gross change, somewhat cloudy and hyperemic, glomeruli swollen and show as reddish or pale greyish dots. Microscopically may be (1) degenerative or (2) proliferative changes in glomeruli.

(1) capillaries congested or hyaline degeneration, epithelium lining Bowman's capsule degenerated and desquamating. Space in capsule is filled with degenerated epithelium, red corpuscles, leucocytes, albumen and granular material.

(2) lining cells show proliferation.

Desquamative papillary nephritis a form of *acute parenchymatous nephritis*.

Acute diffuse nephritis. With parenchymatous changes find also exudation into interstitial tissue and edema and purulent exudate. Parenchyma and supporting tissue about equally affected. Lesions most marked in cortex, as that is the most acive functionally.

Chronic diffuse nephritis. Chronic nephritis, non-indurative, large white kidney.

Combination of interstitial inflam. with degeneration of parenchymatous cells, slow course. Changes like those found in acute and sub-acute stages, but more extreme. Chronic parenchymatous, chronic hemorrhagic and chronic glomerulo-nephritis. Gross. Kidney is enlarged, soft and doughy, capsule peels readily, surface smooth and greyish yellow or greyish white. Sometimes injected veins or minute hemorrhages. Color due to degenerative changes, necrosis and fatty degen., partly to anemia. Color may be uniform or blotchy, patches of congestion alternating with pale areas. Cortex pale uniformly or patchy with occasional areas of congestion. Medulla dull red, congested, in marked contrast with pale cortex. With much hemorrhage is *chronic hem. nephritis.* Microscopically varies somewhat. Fat or soaps in epithelium of glomeruli and tubules and even in endothelium of blood vessels. With this is inflam. edema, leucocyte infiltration in interstitial tissue. In more advanced cases indications of atropy of glomeruli and secreting epithelium of tubules and a little secondary fibrosis.

White spotted kidney of calves—Nephritis fibro-plastica not infrequent. Rare in adult cattle so probably is transitory. Kitt thinks this becomes multiple and diffuse sclerosis which is frequent in adult cattle.

Chronic indurative nephritis—chronic productive nephritis.

Purulent nephritis, infection from blood or bladder.

Diffuse suppurative infect. from blood, micrococcus chiefly.

Capsule contains many small abscesses, many also in cortex, occurs chiefly in pigs and calves.

abscess, chiefly from emboli, as in horses during strangles, in cattle due to purulent pyelo-nephritis, single or several abscesses. Entire kidney may be a fluctuating abscess.

Pyelonephritis from bacteria
Pyelitis
Nephrolithiasis
Parasites Eustrongylus gigas,
larvæ of sclerostomes,
Stephanurus dentatus,
Cysticercus cellulosus,
Echinococcus,
Coccidia
Tumors adenoma, adeno-carcinoma.

OSSEOUS SYSTEM

Rickets. Doubtful as to its nature ; by some thought to be due to infection or intoxication and so to some extent inflammatory ; but it is closely dependent on malnutrition. It may be regarded at present as a disorder of growth. Occurs in the young.

Pathological changes. There is excessive absorption of bone with impairment of the process of calcification. Normal process of lacunar bone absorption is much increased, so solidity of greater part of the skeleton is more or less destroyed. External denser layers become porotic and the trabeculae of the spongy part are attenuated and may even disappear. There is early a formation of new bone that is deficient in lime salts (osteoid tissue), derived from periosteum and marrow, which are extremely vascular. When arising from the marrow the new substance is like the internal callus. The trabeculae from the periosteum are formed in much the same way as the outer callus, are composed of cellular or cellulo-fibrous tissue.

In the periosteum of the long bones cartilage is often laid down, which later is transformed. So bones are covered with a vascular spongy layer, especially at junction with the epiphyses. In extreme cases the original bone becomes porotic. Enchondral ossification is interfered with ; spicules or none at all in zone of calcification. There is always an enlargement of the proliferation zone of cartilage, the columns of cartilage cells longer. Vascular marrow spaces, which gradually encroach on the solid cartilage, are formed. As the disease heals lime salts are deposited. Bones may become harder and denser than normally.

Hyperemia active ; passive, chronic passive hyperemia appears to favor growth of bone, e. g. clubbed fingers and toes in man in chronic pulmonary and cardiac affections.

Hemorrhage is not very uncommon. As a rule blood is readily absorbed in bone

Hemomelanosis of the bones in slaughtered cattle.

Embolism. There are abundant anastomoses in bone, except at the ends of diaphyses.

Thrombosis—not of practical importance.

Retrogressive metamorphoses. The structure of the bones in health is constantly changing. In the young production predominates, the bones increase in size ; in the old resorption is in excess, so bones become smaller and lighter. Lacunar resorption takes place, both in normal and pathological conditions, by means of osteoclasts, situated in the periosteum and bone marrow. These take position on the bony trabeculae and erode their way into the bone forming excavations called Howship's lacunae.

Atrophy. In rapid resorption of bone osteoclasts are increased and lie close together. Surface of bone eroded and rough. When it begins at center *excentric atrophy*, in outside *concentric atrophy.*

In some cases compact bone becomes porous, due to widening of Haversian canals. This known as *osteoporosis.* Atropied bones are light and easily broken. Marrow varies in appearance. Sometimes hyperplastic, resembling lymphoid marrow; fatty or fat may be replaced by semitranslucent, gelatinous material *serous atrophy.*

Atrophy of bone may arise as senile, marantic, from pressure, from disuse or neuropathic disorders. Inflammatory atrophy may follow inflammation.

Death of bone. *Caries* is slow death, in fine particles, is analogous to suppuration of soft tissues; due practically always to inflammation. *Necrosis* may be due to inflammation, interference with circulation, traumatism or chemical or thermal agents. Ordinarily the necrotic part of bone becomes separated from the healthy part by zone of inflammation where resorption and exfoliation are actively going on. Such a piece is called a *sequestrum.*

Halisteresis. Organic substances remain unaltered, but lime salts are diminished, so bone becomes soft and yielding. May be in restricted area or may be general. When more extended is known as *osteomalacia.* In general there is decalcification of old bone with a tendency to form new which remains imperfectly calcified. Decalcification begins at the periphery of bone trabeculae and gradually goes deeper. It occurs in adults, may be in young. It is not an anomaly of growth.

Inflammation affects the vascular structures chiefly i. e., the periosteum and bone marrow, *periostitis* and *osteomyelitis.* Compact bone may be involved secondarily in either condition, *ostitis* usually in the form of erosion and disintegration.

Periostitis is the most frequent in the domesticated animals. Due to local or systematic causes, usually traumatic, may be infectious or toxic. Results vary. Mild inflammations that are treated early and efficiently may heal without noticable effect.

Often there are serious results (a) disintegration, (b) proliferation; may be both processes together.

(a) necrosis is a marked feature in many acute and chronic inflammations. It may be slow eroding (*caries*), or exfoliation of larger masses (*sequestration*). Bone may become *porotic*, owing to lacunar resorption, resulting from action of granulation tissue in the medullary spaces, (*rarefying ostitis*).

(b) Bone formation may be stimulated, e. g. near abscesses, sequestra, injuries, etc. The bone becomes harder and denser by deposit of more bone on the trabeculae (*osteosclerosis*)).

Acute simple periostitis, a simple aseptic inflam. Cellular infiltration, tissue swollen, reddened, with scattered hemorrhagic spots; may be hemorrhagic periostitis when hemorrhage is marked feature. Acute simple periostitis is present in fractures.

Chronic fibro-plastic periostitis. In this form the tissue of the periosteum is increased. Is longer continued than the above. With repeated trauma; may be chronic from the beginning. Usually passes into the next form.

Chronic ossifying periostitis. Frequent in the horse and cow. There is an increase of the inner layer, osteoblasts. May be diffuse or local.

Exostoses or *osteophytes*, *hyperostosis*, *paraostoses* in connective tissue near bone not joined to bone.

Suppurative periostitis, due to infection usually from external injury; may be hematogenous, then belong to group of septicemic affections; may extend from marrow or from neighboring tissues (skin). Is severe, involves neighboring parts. Characterized by suppuration and necrosis. In the acute form the affected region swollen, tense, reddened and intensely painful. May extend to bone marrow (*osteomyelitis*) Sometimes epiphyses and joints involved. In mild cases may terminate without much disturbance; but owing to interference to the circulation by pressure of exudates, larger or smaller portions of bone necrose. May be in center or on surface. Surface where periosteum has been lifted from bone. When bone has been splintered, fractured or crushed, it is inflamed; may readily heal. If microorganisms are circulating they may localize in the injured part and condition similar to infective osteomyelitis and periostitis be produced. In a compound fracture may be infection from without.

Ostitis and osteomyelitis, inflammation of bone and marrow.

Rarefying ostitis, bone becomes spongy, due to action of granulation tissue in the medullary spaces; absorption in lacunae and Haversian canals.

Osteosclerosis.

Spavin and ringbone.

Suppurative ostitis and osteomyelitis.

Actinomycosis.

Tuberculosis.

Echinococcus, the only parasite found in the bones.

Progressive metamorphoses. In adults waste and repair are balanced. Sometimes waste may exceed.

Hyperplasia, cause sometimes obscure.

a. congenital and primary e. g. gigantism.
b. acquired and secondary e. g. after fracture and in some cases of inflammation; experimentally by driving ivory pegs into growing ends.
c. In some cases due to chemical substances circulating in the blood e. g. P, As. stimulate bone production experimentally.
d. associated with disorders of internal secretion e. g. acromegaly.

Prolonged passive congestion seems to favor overgrowth e. g. clubbed fingers of those with chronic pulmonary and cardiac affections.

It may be compensatory e. g. after fracture of tibia, fibula increases in size.

Overproduction may occur in several ways—

hyperostosis, when bone is enlarged in whole or part,
osteosclerosis, when density is increased,
osteophytes or *exostoses*, local outgrowths,
enostoses, local growths within spongiosa.

Fracture, simple or uncomplicated; compound or complicated.

Callus, after fracture repair begins in periosteum and marrow. With injury is tearing and bruising of soft tissues with effusion of blood; moderate inflammation with exudation of fluid and infiltration of leucocytes. This subsides in 5–6 days. About the second day cells of periosteum and marrow show signs of proliferation. This increases greatly in next few days and endothelium of blood vessels joins in, so about 4th day the osteoblastic layer of periosteum is changed into a vascular germinal tissue. After 4th day germinal layer begins to differentiate into chondroid and osteoid tissue which is rapidly converted into bone. After a week ends of fractured bone are embedded in large number of young osteophytes and bone spicules. So about the external injury is produced a spindle shaped sheath, the *external callus*. A continuation of this between the broken ends is the *intermediary callus*.

A callus is formed within the cavity of the bone (if a hollow bone) constituting the *internal callus*. This is produced by osteoblasts grouped in masses and are transformed into osteoid and finally into osseous tissue.

In two or three weeks the ends of bone are united. Amount of callus differs much depending on individual, condition of bone, nature of fracture and amount of deformity. With formation of new tissue, is resorption. Broken ends become rounded, splinters are resorbed. Callus at end of 6th or 7th week is rather soft and porous. The bony tissue becomes harder, so the *permanent* or *definitive callus* is formed. After months or years the permanent callus is modified further.

Tumors. They primarily belong to the connective tissue group. They arise from the periosteum, marrow or cartilage. Fibroma, chondroma, myeloma, osteoma, sarcoma, osteoid sarcoma, melanosarcoma.

JOINTS

Structure and function.

Hyperemia, *active* in early stages of inflammation, *passive* under same conditions as elsewhere.

Hemorrhage, usually due to traumatism. Small effusions are rapidly absorbed; larger ones may be partly absorbed, the remainder replaced by fibrous connective tissue.

Degeneration, may affect articular surface or synovial membranes. Fatty, hyaline, amyloid and mucoid.

Necrosis and caries.

Pigmentation.

Uric acid deposit, especially occuring in birds.

Distortion.

Luxation and subluxation.

Deformities of vertebral column,—lordosis, kyphosis, scoliosis, wry neck.

Inflammation, acute inflammation may involve first the lining membrane (*acute synovitis*), the cartilage (*chondritis*); with severe inflammation bones and soft tissues are usually also involved (*ostitis and periarthritis*).

Acute arthritis and *synovitis* arise from traumatism or by extension from neighboring tissues; some cases are hematogenic e. g. navel ill, acute articular rheumatism. In early stages of severe and in milder forms the synovial membrane is hyperemic and swollen, especially about the folds. There may be small hemorrhages and exudation of thin yellowish fluid containing a few floculi of fibrin (*serous synovitis*). In more severe cases the exudate contains more fibrin (*serofibrinous synovitis*); in some cases exudate may be mostly fibrin with a little fluid (*fibrinous synovitis*). Exudate may become purulent or may be purulent from the beginning (*purulent synovitis*, *empyema articuli*). In this case the synovial membrane is swollen, infiltrated and covered with fibrinopurulent or purulent exudate, the cavity containing turbid fluid. In the most severe forms the synovial membrane may be partly destroyed and cartilage may undergo fatty degeneration, fibrillation and finally necrosis. The joint may become disorganized. Process frequently extends to bones.

Acute synovitis and arthritis often heal leaving little or no trace, or may pass into a chronic condition. More or less fibrous proliferation often occurs during repair. Osteophytes forming about the joint, ankylosis sometimes results.

Chronic arthritis

Chronic serous arthritis arises from the acute or may be chronic from the beginning. This form is common in horses and cattle in fetlock, tarsal and knee joints. "Galls"

Chronic suppurative, empyema of the joint.

Chronic arthritis

- (a) *chronic serous proliferating synovitis*;
- (b) *arthritis ankylopoetica*, *ankylotica*;
- (c) *arthritis deformans*, two forms the proliferative and the degenerative. *Osteoarthritis intertarsalis ankylopoetica ossificans* (spavin) and ringbone. A disease primarily of bone, a rarefying ostitis, which may extend to involve articular cartilage, or may extend to periosteum or may involve both. Destruction of cartilage, ulcerative arthritis, with proliferation of cartilage and other tissue, formation of exostoses, ossifying periostitis, ankylosis, osteosclerosis, etc.

Arthritis uratica s.urica, gout, occurs in birds and man, sometimes in dogs.

SKIN.

ucture and function.

Anomalies

hypotrichosis in swine

Hyperemia shows best in unpigmented areas. *Active* in inflammation, *erythema*, in vasomotor disturbances, excessive heat and cold, mechanical and chemical irritants. *Passive* in conditions favoring stasis, may be diffuse, *cyanosis*, or in areas, *livido* (*lividity*), after death, postmortem lividity, local passive hyperemia may be due to pressure of tumors or of inflammatory products on veins.

Edema

Hemorrhage, generally traumatic, also in infectious diseases, petechiae, ecchymoses, vibices, purpura papulosa, hematoma, hematidrosis, suggilation.

Emphysema

Anemia, may be general or local.

Atrophy

Necrosis, ulceration, gangrene.

Abnormal pigmentation

Melanosis maculosa in calves.

Inflammation *dermatitis*. Inflammations of the skin present much-varied appearances according to cause, course and anatomical changes. Different causes produce similar changes. No satisfactory classification yet made. Is generally due to external causes, mechanical injuries, chemicals, heat, cold, light, local infections, vegetable and animal parasites. Sometimes dermatitis seems to be neuropathic (e. g. urticaria prurigo) and some forms are of unknown or doubtful origin. Some inflammations of the skin are secondary, accompanying general infectious diseases, or are due to the action of toxic substances taken internally. These hematogenic affections of the skin are commonly called *exanthemata*. Diffuse redness and swelling is called *erythema*. Color is bright, not the dull livitidity of passive hyperemia; disappears momentarily on pressure. Locally raised areas from minute to size of pea due to infiltration in the skin called *papules*; larger elevations to size of hazelnut called *nodules* or *nodes*. *Wheals* are broad, flattened, fairly well-defined elevations which appear and disappear rapidly, color dull reddish, larger ones may have whitish center. Microscopically in these mild forms of dermatitis, one finds infiltration with serum and leucocytes, sometimes red corpuscles also in tissue. Epithelium is but little changed, may be swollen. There may be slight proliferation. With greater amount of serous exudation, local collections of fluid occur, *blisters*, *blebs* or *vesicles*. Fluid clear, without cells. May be somewhat turbid from leucocytes or reddish from blood (*blood* blisters). Exudate turbid, whitish (purulent), *pustule*. When pustule dries forms *scab* or *crust*.

A mild acute dermatitis, may pass quickly, leaving little or no trace. Frequently even after erythema superficial epithelium desquamates. Pigmentation also common sequel. If papilary layer or corium is damaged, scar results. In chronic dermatitis atrophy and hypertrophy may be combined. Superficial dermatitis which resembles catarrhal inflammation of mucous membrane is ordinarily called *eczema*.

One of the simplest forms of dermatitis is due to exposure to sun (*sunburn*). Blood vessels relaxed, active hyperemia and slight exudation. After process has subsided there is desquamation and more or less pigmentation. Inflammation due to burning in mildest form is similar (*burn of the first degree*). With higher temperature or more prolonged exposure, injury is more severe. There is considerable exudation, epidermis is raised into vesicles (*burn of the second degree*). With loss of substance of cutis (*burn of the third degree*). If charred (*burn of the fourth degree*). Burns of the second degree heal by simple regeneration of the epithelium if there is no infection. With loss of substance healing takes places by granulation with formation of scar. Dermatitis of frost bite is very similar. There may be different degrees, erythema with swelling and later desquamation or formation of vesicles or gangrene.

Exudative dermatitis is a common form and exhibits a great variety of appearances. Sometimes the lesions are restricted, sometimes diffuse. The skin is reddened and swollen. It may be covered with papules, vesicles, pustules or crusts. The affected part may itch. It may desquamate. In chronic cases the skin becomes thickened. A number of names are in use to distinguish different stages of exudative dermatitis and differences in the kind of exudate. One of the mildest *dermatitis papulosa*, characterized by small nodular swellings in the skin. In somewhat more severe cases vesicules are produced, *dermatitis vesiculosa*. With considerable areas of skin painful, red and swollen, *dermatitis erythematosa*. If blisters formed in this inflamed area, later becoming pustules, *dermatitis pustulosa*. When considerable patches of epidermis are lost, the underlying surface presents a dusky red appearance, *dermatitis rubra.* Often the serous or purulent exudate dries up on the surface into yellowish crusts, *dermatitis crustosa.* Under the crusts pus may accumulate, *dermatitis inpetiginosa.* In other cases the epithelium may proliferate beneath the scabs so that the surface appears reddened, thickened and scurfy, *dermatitis squamosa.* When somewhat larger pustules covered with crusts occur, it is often called *impetigo.* Histolog· in acute inflammation, congestion of blood and lymph vessels in corium, exudation of plasma and diapedesis of leucocytes. Connective tissue fibers are swollen and compressed and there is some proliferation of the connective tissue cells. In the milder form process confined mainly to papillary regions and upper layers of corium, but in more severe cases extends to subcutem. Cells of rete swollen, effusion of fluid with wandering cells. Rete cells may undergo liquefacation necrosis with formation of cavities. When exudation marked, vesicles or bullae may form between rete and horny layer. In pustular variety many leucocytes. Desquamation of superficial epidermis takes place often and is a characteristic change in dermtitis squamosa and rubra.

Dermatitis phlegmonosa, phlegmon of the skin ; panaritium.

Suppurative dermatitis, abscesses of the skin, ulcers, fistulae.

Dermatitis gangraenosa, gangrene of the skin.

Chronic productive, *dermatitis*, restricted as on lower part of foot of horse, or diffuse.

Tuberculosis

Glanders

Epizootic lymphangitis

Vaccinia and Variola

Epithelioma contagiosum

Actinomycosis

Botryomycosis

Parasites, long list

flies, lice, ticks, acari, worms.

Herpes tonsurans, favus. (See parasitologies).

Progressive changes, hypertrophy, cornu cutaneum, callus, elephantiasis.

Tumors. Fibroma, papilloma, lipoma, chondroma, angioma, adenoma, adenocystoma, carcinoma, sarcoma (rare), Hodgkin's disease, melanoma (frequent in horse).

References.

Adami and Nicholls, pp. 907-977.

Kitt, pp. 134-243.

MAMMARY GLANDS

Structure and function.

Hyperemia, physiological, ante partum and post partum.

Hemorrhage, suggillation, hematoma.

Edema, congestive, stagnation, collateral and inflammatory.

Concretions, in cow and goat.

Cysts.

Inflammations that arise naturally are due to infection. Several species of bacteria culpable. Enter through milk ducts (teat). May enter through the blood.

Acute catarrhal galactophoritis, inflammation of the milk ducts, due to infection through teats ; may be manifest only on examination of sediment or microscopic examination of the organ.

Acute parenchymatous mastitis. Inflammatory edema often over the entire gland, gland sometimes swollen to double its normal size. On section, color red, red gray or even dark wine red ; milk ducts filled with flocculent whey, may have fibrin adhering to walls, fluid may be serous, bloody.

Suppurative mastitis. Purulent exudate into ducts, the secretion may be rich in leucocytes. Process may go on to abcess formation.

Gangrenous mastitis, more frequent in sheep, fatal in twenty-four hours to five days.

Chronic productive mastitis, increase in connective tissue, usually follows acute inflammation.

Thelitis, inflammation of the teat, nipple, superficial is inflammation of the skin.

Thelitis mortificans, in cow.

Chronic productive thelitis, in inside, production of connective tissue leading to obstruction.

Infectious mastitis.

Glanders.

Tuberculosis.

Actinomycosis.

Botryomycosis.

Animal parasites.

Echinococcus in cow.

Tumors. Fibroma, adenofibroma, enchondroma, osteoma, angioma, sarcoma, and carcinoma.

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LeBlanc, "The Diseases of the Mammary Glands of the Domesticated Animals", translated by Nunn, 1904.